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Gastrointestinal Bleeding

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Associate Clinical Professor of Medicine at Harvard Medical School, has been affiliated with this school since 1940. During 1949-52, he served as Clinical Professor of Medicine at Tufts College, School of Medicine, and was Lecturer on Medicine during that period. During World War II, he was assigned in the Southwest Pacific Theatre and, because of the experience there in infections and other disorders of the gastrointestinal system, joined the Veterans Administration on release from active duty. Since March, 1946, he has served as Chief of the Medical Service at the West Roxbury Veterans Administration Hospital. Owing to the high incidence of gastrointestinal disorders in the veteran group, Dr. Warthin has had an unusual opportunity to study problems of gastrointestinal ulceration and its complications, hepatic disorders and latent tropical diseases of the gastrointestinal tract.

THE MOUNTING incidence of bleeding from the gastrointestinal tract during the last two decades is reflected clearly in the rising volume of papers, articles and lectures on this subject. During World War II, and especially since then, hospital admissions have included an increasing percentage of such patients. Reports have been published from various clinics, including those whose patient populations arise from predominantly upper or lower economic brackets. There seems little doubt that a higher incidence occurs in the latter group and that a more vigorous diagnostic and therapeutic-surgical approach stems from these clinics. It is recognized that this is a generalization and therefore open to criticism, but one can often foretell policies of management if one knows that the author is using material gleaned from his experience at a charity hospital or from a 20 year study of his own private patients. Since these differences are particularly true in the management of upper gastrointestinal bleeding, they will be dealt with in more detail below. It is my desire here merely to stress the fact that controversy does exist, but that the practitioner

can largely resolve this difference if he adjusts his policy of management to the type of patient and to his own local medical and hospital situation.

Some hospitals have reported that as many as 1.7% of all their admissions are for intestinal tract bleeding, not including those obviously due to oral or anal causes. It seems likely that at least 50,000 patients with peptic ulcer require hospitalization for hemorrhage each year. More specific figures of admission rates for bleeding are not available. These rates certainly average around 1% in urban areas and possibly one-half that in more rural communities.

UPPER GASTROINTESTINAL HEMORRHAGE

One point in the management of such bleeding is worthy of special stress—the "team" policy. The day when a single individual can adequately handle gastrointestinal bleeding patients in a modern hospital has passed. This is especially true if massive bleeding is occurring. In these instances the assistance of a surgeon, the clinical laboratory, a radiologist and operating room personnel may spell the difference between life and death. As has been aptly stated—bleeding ulcer patients are best managed by eager internists together with reluctant surgeons. Multiple therapeutic possibilities have complicated, rather than eased, the clinician's lot. Indeed, the refinement of old tools and discovery of new ones are somewhat inevitably followed by a tendency to use them, resulting in a more aggressive management. This, under the proper team conditions, will reduce mortality and disability from bleeding. But it does not cast discredit on the results of older methods, which may still be better effected in the small community hospital where patient care is a one-man task. In the latter circumstance, concentration on the conservative feeding and infrequent transfusion regimens of the 1930's will be best. With this policy, these mortality rates can be as low as 6.5% and average 10%. Conversely, a purely surgical approach has resulted in an even higher mortality rate. The argument that the bleeding ulcer patient will eventually come to surgery, so "why not now," has no place in altering the management of the active bleeder.

The vast majority of hospitals, however, contain a well trained

staff of internist, surgeon, clinical pathologist and radiologist supported by an adequate blood bank and operating room personnel. A team of these doctors, aware of the varied problems in management of gastrointestinal bleeding, and trained in the necessary diagnostic and therapeutic procedures applicable in such cases, can accurately pinpoint the source of hemorrhage, if it be not known, reduce the fatalities from bleeding peptic ulcer to less than 2% and, perhaps even more strikingly, save the individual with an unusual source of his bleeding. Such a team should be alert to select early in the course of the bleeding episode those patients most apt to continue or recurrently hemorrhage. Some of these patients would die if medical therapy were continued, but with early operation the surgical mortality will be that of elective gastrectomy, approximately 2%.

General Management of Upper Gastrointestinal Bleeding

The general management of such patients will be discussed first, followed by specific comments regarding individual lesions. It is important to establish a few definitions at this moment. "Upper" gastrointestinal hemorrhage includes bleeding from lesions located from the oropharynx to the upper jejunum, beyond the ligament of Treitz. Bleeding from any of these locations can result in hematemesis, a more rapid and massive hemorrhage being necessary from the duodenum and beyond than from more proximal sources. Bright red blood with or without clots is most characteristic of bleeding from the esophagus or stomach, but bleeding from lesions as low as the ampulla of Vater can certainly mimic this at times. The presence of hematemesis accurately labels the bleeding as arising in the "upper" tract. Failing this, and with melena alone, one may be forced on occasion to determine the approximate site of origin of the changed blood. This can safely be done by passing a soft stomach tube and aspirating the gastric contents, after ruling out the likelihood of esophageal varices. If this procedure fails to produce coffee-ground guaiac-positive material, a white string may be swallowed, as recommended by Einhorn. The patient is kept on his right side, and after four or more hours the string is withdrawn and the

distance from mouth to area of blood measured. This simple procedure is not completely reliable, as minor or insignificant bleeding above the major source may obscure the actual site. In my experience, small intestinal lesions have more often been located by operation or by passage of a Miller-Abbott tube, with continued aspiration for changed blood as the tube moves down the intestine.

Most of the comments will refer to "massive" hemorrhage, since mild or moderate bleeding requires only simple treatment and is accompanied by almost no direct mortality. It should be realized that severe bleeding can occur in debilitated individuals without any such external manifestation as melena or hematemesis. Rarely, it occurs so rapidly that an individual may be in shock with normal hematocrit and hemoglobin values although his blood volume is greatly reduced. Hematemesis is a very suggestive sign that massive bleeding is occurring, particularly in patients with duodenal ulcer. A massive hemorrhage can be said to have occurred if the hemoglobin content falls below 8.0 Gm per 100 cc, the red blood cell count below 3,000,000, the hematocrit reading below 30%, or at least 1,000 cc of blood is required immediately to correct a state of blood volume insufficiency.

All patients with upper gastrointestinal hemorrhage should be admitted to the medical service, but our surgical colleagues should be promptly notified of such admission and see each patient with us within four hours. An orderly, sympathetic and reassuring attitude of the physician is immediately helpful for the patient. Our interests are directed toward five spheres: (1) treatment of shock and restoration of adequate circulating blood volume; (2) medical control of bleeding; (3) investigation of the source of bleeding; (4) study of the patient for evidence of significant complications including systems other than the gastrointestinal tract; (5) estimation of rate and manner of bleeding.

1. Treatment of Shock.—An infusion of 5% glucose in normal saline should be promptly begun and the patient's blood cross-matched. Five hundred mg of vitamin C and approximately 60 mg of vitamin K are added to this infusion. The common signs of shock should be sought. It has been recognized for many years that hematemesis is one of the cardinal symptoms of the more major hemorrhages. If this symptom is present, and red

rather than tarry stools are passed, one can safely assume that one or more liters of blood have been lost and plan replacement accordingly. Elderly patients with heart disease may lack tachycardia. If previously hypertensive, they may appear to have an adequate blood pressure, whereas, in reality, insufficiency of brain, heart or kidneys may be impending. *I believe that blood transfusions are the essential backbone of therapy and should be administered as soon as cross-matching can be performed and at a speed and volume necessary to maintain the hemoglobin content above 10.0 Gm per 100 cc and the red cell count above 3,500,000.* This insures an adequate blood volume reserve so that, should bleeding recur, irreversible shock will not appear. Not infrequently, two or more extremities are needed to infuse blood rapidly enough to keep ahead of the shock and continuing bleeding. If possible, 3 L of blood should be cross-matched as a reserve, should operation become necessary. The pulse, blood pressure, stools, vomitus and transfusions should be all charted together to present a complete graphic picture of the patient's course. The urine output should be measured every 24 hours. It is worth emphasizing that when a patient stops bleeding after repeated or prolonged hemorrhage and when hemodilution has taken place, oliguria secondary to decreased renal blood flow or other factors may produce a situation wherein the patient has an excessive blood volume although he is still fairly anemic. Should further transfusions be administered, an elderly patient with heart disease will respond with a rising pulse. This may be mistaken as evidence of further bleeding and the flow of blood transfusion accelerated, thus producing pulmonary edema. A glance at the simultaneously rising pulse, blood pressure, full neck veins and empty urine bottle tell the true story. Discontinuance of the transfusion will rapidly correct the dyspnea. Elevations of pulse associated with fever from mild pyrogenic reactions should not be confused with additional bleeding.

2. MEDICAL CONTROL OF BLEEDING.—It has been repeatedly demonstrated that a program of *feeding* is very effective in the moderately bleeding ulcer patient, and such a routine should be followed initially since 75% of all bleeders have ulcers. However, the patient who is vomiting frequently will not retain food. Since the blood in his stomach is a good buffering agent and he might

possibly be a candidate for surgery, food should be withheld until several, usually four, hours have elapsed since the last hematemesis. Small amounts of milk, gelatin or custard are then fed hourly, day and night. In the proved ulcer patient, give 2.0 Gm of calcium carbonate with or without 0.1 Gm of magnesium oxide between these feedings. The dosage of magnesium oxide can be increased if constipation is present or omitted if diarrhea appears. As soon as bleeding has been halted for 48 hours, bland soft solids and two-hourly feedings may be begun. Aluminum hydroxide gel and magnesium trisilicate combine with blood to produce severe fecal impactions, so they are best omitted until bleeding has ceased. Then they can be substituted for the calcium carbonate. Vitamins C and K are arbitrarily and not too scientifically employed in large doses in the initial intravenous solution and daily thereafter.

Sedation of the ulcer bleeder, except the elderly, must be effective. This is best accomplished by combinations of 50–75 mg of Demerol subcutaneously, alternating every four or six hours with either 120 mg of sodium phenobarbital or 0.2 Gm of sodium Amytal intramuscularly. Sodium Amytal is reported to decrease gastric secretion and acidity. Morphine is very apt to evoke vomiting and should rarely be used as it causes gastric hypersecretion and hypermotility. The patient with cirrhosis should, of course, receive only a small initial dose of sedative. Effective sedation, however, is a most important factor in preventing blood pressure elevations based on anxiety or emotion. This avoids the frequently criticized hazard of transfusion therapy, namely, extrusion of the fresh clot. A young bleeder should be kept in a drowsy state, awakening readily when feedings are given or blood pressure is taken but immediately returning to slumber. Elderly patients cannot safely be sedated to this point because of pulmonary hypostasis. Fortunately, phlebothrombosis seems to be a rare occurrence in these patients and need not be a cause for concern.

Atropine in 0.7 mg doses is injected subcutaneously every six hours in the young bleeder. It should not be given to the elderly or the alcoholic, as it is apt to initiate a toxic psychosis in the latter, and its drying effect on respiratory secretion adds another hazard, beyond the induction of glaucoma, in the former. Buffered thrombin is recommended only for patients with known

gastritis or if other measures have not been effectual in stopping ulcer bleeding. I have used this therapy in over 25 patients and have not been impressed with a frequent coincidental cessation of hemorrhage.

3. Source of bleeding.—If an aggressive approach to the bleeding patient is to be followed, an accurate determination of the source of the bleeding is essential. In private practice, approximately 75% of these patients arrive at the hospital with the source fairly adequately tagged, having been previously determined by x-ray. Much less certainty pertains to the urban clinic patient, of whom less than 50% are admitted with appropriate labels already attached. In the latter group in particular, multiple sources may be suggested by the history and physical examination. Combinations of hiatus hernia and peptic ulcer or more commonly peptic ulcer and cirrhosis with or without varices arise as nightmares of diagnostic delirium. In cirrhotics, the incidence of peptic ulcer is three or four times above normal. In my experience, 15% of massive bleeders have had two or more possible sources for their bleeding. Yet the determination of the source of hemorrhage has become increasingly important now that surgical treatment can be effected not only for the ulcer patient but for control of certain esophageal lesions as well.

The problem of patients with an undetermined origin of bleeding is common, varying from 10 to 25% in most clinics. The uncertainty as to whether the bleeding comes from a benign ulcer or a malignant tumor has an adverse effect on both patient and physician. It is my thesis that, in appropriate circumstances, safe diagnostic studies can be made during the period of active bleeding. This investigation will reduce by 50% the group of bleeders from an undetermined source.

Table 1 indicates our results with 246 patients with massive bleeding in a five year study at a Veterans Administration hospital, and the outcome as far as survival is concerned (1). As might be expected, 75% were patients with peptic ulcer. The ratio of gastric to duodenal ulcers is a little lower than might have been expected—1:8 instead of 1:6. Three of the gastric ulcer deaths occurred early in the series and might have been prevented by an aggressive diagnostic and surgical approach. Thus, my current policies might have produced a better mortality than the 3.2%

that occurred. Thirty-two emergency or urgent operations were performed during the study with a single death. The rather generous number of cirrhotics in the series reflects the all-male population of a V.A. hospital. The extremely bad results with bleeding varices will be discussed later. In the first two years of the study, the source-unknown group comprised 15% of the total, but, again, a more aggressive diagnostic approach cut this in half, and the average for the five years was 10%.

The *history* must be carefully checked for previous or recent ulcer symptoms. A history of alcoholism or jaundice cannot be

TABLE 1.—Causes of Massive Bleeding in 246 Patients Observed during Five Years in a Veteran's Administration Hospital*

Diagnosis	No. of Patients	Deaths
Peptic ulcer	184	6
Duodenal ulcer	161	2
Gastric ulcer	20	4
Marginal ulcer	3	0
Esophageal varices	20	16
Gastritis	13	0
Miscellaneous†	4	2
Source unknown	25	0

*From Warthin *et al.*: Ann. Int. Med. 39:241–253, 1953.
†Carcinoma, benign tumor, cirsoid aneurysm, uremia.

taken lightly. A recent x-ray diagnosis of ulcer was found very reliable in indicating that lesion as the culprit. The physical examination, if normal except for epigastric tenderness, suggests ulcer, but the presence of a large liver and numerous spider telangiectasias does not rule out an ulcer. Sixteen per cent of our cirrhotic patients had a peptic ulcer as well. To my surprise and dismay, I found that my ability to diagnose clinically the source of bleeding in the new patient in the older age group was only approximately 70% accurate. Since these are the patients in whom our special interest lies, I resort to the *emergency gastrointestinal x-ray examination* of Hampton, as modified by Schatzki. Between 1946 and 1950 I employed this maneuver only when these patients had continued to bleed for 36–48 hours. As a result of this policy, three patients with gastric ulcer died needlessly of frank exsanguination. Such deaths, entirely preventable, as are all exsanguinations, made me change my policy to a program of

emergency gastrointestinal x-ray examination of all new patients over 50 years of age as soon as they have been brought out of shock. The films obtained are then reviewed by all members of the team concerned with these bleeding patients.

Between 1946 and 1950 we performed 100 gastrointestinal x-rays within the first 72 hours of admission. The results are given

TABLE 2.—Emergency Gastrointestinal Roentgen Examinations

1946–51 (within 72 hours of admission)	100 patients
1951–53 (within 12 hours of admission)	75 patients
No diagnosis established	28 (16%)
Incorrect diagnosis	24 (14%)
Procedure not completed	7 (4%)
Possible recurrent bleeding	5 (3%)

in Table 2. In the succeeding 18 months, 75 such examinations were made within 12 hours of admission with equal *freedom from renewal* of bleeding as long as the patient was not in shock, was only turned and never heavily palpated. Inability to swallow is an obvious contraindication to such examination. The results reflect to a large extent the radiologist's interest and willingness to make a careful study in trying circumstances. Some radiologists are not so inclined, and it is my feeling that this attitude results in an unsatisfactory examination. In these circumstances, it should not be attempted. Most errors were related to duodenal lesions, but a few significant gastric lesions were not demonstrated. Esophageal varices were well demonstrated with thick or thin barium. I am unaware of ever having missed them when they were indeed the source of bleeding. Spot films of the esophagus should always be taken, even though on fluoroscopy it appears normal, because small varices or tumors may be demonstrated by this technique. It might be mentioned at this point that patients in incipient delirium tremens are apt to misinterpret in various ways the satanic appearance of the radiologist with his goggles, hard table and flashing red lights of the fluoroscopic room. For this and the obvious reason that the patient must be constantly observed, the physician must always be present when the examination is done. Gastric ulcers are usually demonstrated best after a single swallow of Rugar barium which, being heavier than blood clot, slips around on the dependent surface of the stomach

until the proper position is obtained when it puddles amid the shadow contrast.

Patient A, age 76, had begun to bleed 12 hours before hospitalization. Vague epigastric distress was noted for several weeks before the sudden onset of hematemesis and melena. On admission to the hospital, transfusion of 1,500 cc. of whole blood restored his circulating blood volume, and an emergency G–I series was performed four hours later by a disinterested radiologist, using a glassful of thin barium. No source for the bleeding was found. Eight hours after this x-ray examination, massive bleeding recurred. A repeat study was then made by the resident in radiology, using 2 teaspoonsful of thick barium. The patient was gently turned from side to side. A large gastric ulcer at the angulus of the lesser curvature was found without too much difficulty by this technique. It is seen in Figure 1, *A* (p. 17) framed by the barium in the colon from the previous x-ray examination.

Spot films (Fig. 1, *B*) taken at various angles illustrate the size of the ulcer and its ease of demonstration if the stomach is not overloaded with contrast medium. The patient recovered uneventfully after an emergency operation.

Duodenal ulcer craters are harder to demonstrate, but the deformity of the duodenal cap is easily seen.

Another advantage of early roentgen examination is the reduction of the number of "unknown source of bleeding" patients by fully half—for our hospital from 15% in the first three years of study to 7% during the next two. I have repeatedly seen rapid healing of small gastric or duodenal ulcers within 12 days. I agree with Zamcheck and his associates (2) that early x-ray examination of the shock-free patient is thoroughly safe and in interested hands is a most valuable aid in diagnosis of the origin of severe bleeding.

The development of the *Sengstaken double balloon tube* (see p. 21) has also proved to be of great assistance in diagnosis as well as in treatment. When properly positioned, it always stops massive bleeding from esophageal varices. It may be utilized immediately in the cirrhotic patient with exsanguinating hematemesis, in whom its insertion will be followed by cessation of bleeding, should varices be the source. This procedure is much simpler than esophagoscopy, which I have found unnecessary. If a skilful endoscopist is available, better results in diagnosis may be obtained by the latter examination. Conversely, when two sources of bleeding are possible, e.g., esophageal varices and peptic ulcer, one may pass the tube and inflate both balloons. If bleeding persists, and the balloons are shown by x-ray to be in their proper position,

there is presumptive evidence that bleeding is coming from the ulcer and not the varices. Thin barium can also be introduced into the stomach via the tube and an ulcer demonstrated, if x-ray studies have not previously been made.

If melena alone has been present and no source of bleeding found by emergency x-ray examination, the passage of a soft tube into the stomach and duodenum and aspiration of the contents has proved helpful in establishing the presence of the lesion above or below the ligament of Treitz. The existence of a tumor of the small intestine must then be brought to mind if guaiac-negative aspirate is obtained.

4. Complications.—Study of the patient for significant complications, including systems other than the gastrointestinal tract, is important. These and the rate and manner of bleeding are the determinants by which the future course of treatment—conservative medical or surgical—is decided. The significant information about those patients likely to fare badly was determined by a review of the records of those who died or required an emergency operation to halt exsanguination. It was immediately apparent that half of our *gastric ulcer* patients fell into this category. Others have concurred in this experience and pointed out that erosion in the right or left gastric artery exposes larger vessels than do ulcers in the duodenum. It can be said fairly conclusively that in the presence of a gastric ulcer, hematemesis and recurrence of bleeding after hospitalization, prompt gastrectomy is advisable.

We have also noted a high incidence of massive bleeding among our patients with postbulbar or second portion duodenal ulcers. Here again, recurrent bleeding after 24–36 hours of hospital therapy should be an indication for early operation. Such ulcers are particularly hazardous because the posterior duodenopancreatic artery lies immediately adjacent to this area and is easily eroded, with or without penetration of the pancreas itself.

Much has been written about the significance of age as an indication for operation. We do not believe that chronological age is significant, although physiologic antiquity may be. Specifically, this means vascular, renal, hepatic or metabolic senescence, which may jeopardize an important organ or system during periods of recurrent hypotension or anoxemia. It is known that peptic ulcers

in diabetics often heal less rapidly than those in nondiabetics, presumably owing to altered tissue factors or vascular disease. Peptic ulcers with such vascular disorders as Buerger's disease and polyarteritis nodosa are highly resistant to medical treatment and, if bleeding, will usually continue to do so.

Cirrhosis of the liver combines poor tissue repair elements and frequent portal hypertension as serious complications of medical treatment. Recurrent hemorrhage is a frequent precursor of hepatic coma, so that an early aggressive approach is necessary in cirrhotics. I have often regretted that I failed to push surgery in such a situation, but only rarely regretted having done so. Chronic renal disease, like cirrhosis, combines poor tissue repair and bleeding tendencies to handicap medical therapy. The careful differentiation between the irreversible uremia of far-advanced nephritis and the pre-renal azotemia of mild renal disease and severe bleeding is essential. Surgery is useless in the former, but may be lifesaving in the latter.

It is extremely important, therefore, that every new bleeding patient be thoroughly screened for some complicating disorder. While shock is being treated, a thorough history is taken and a complete but gentle physical examination done. A urinalysis will determine the presence or absence of diabetes or nephritis. Particular attention is paid to the specific gravity as an aid in differentiation between pre-renal azotemia and advanced nephritis. A blood smear will show the presence of abnormal cells of leukemia or the insufficient platelets of thrombocytopenic purpura. The prothrombin time, if prolonged, suggests the presence of a hepatic disorder and is an indication for a bromsulfalein test. An electrocardiogram should always be taken. The finding of an ischemic pattern indicates the especial need for prevention of further anoxemia by transfusions and oxygen therapy. Minor prolongation of the Q-T interval or T wave inversions are not serious. Heart disease is not a contraindication for early surgery. Whenever an emergency G–I series is made, a chest x-ray should be taken for evidence of tuberculosis or tumor. Again it should be emphasized that age itself is not a stimulant for early surgery, but the vascular problems so frequently accompanying this involution can be. Since blindness, hemiplegia, coronary occlusion or ischemia, acute renal failure and peripheral vascular disorders

may all be precipitated by uncontrolled hemorrhage and reduced blood volume, careful estimation of trends or changes in these organs should be watched for. I have observed transient hemiplegia develop in a bleeder shortly after arrival at the hospital, noted its disappearance on transfusion therapy and return with recurrent hemorrhage. During the operation that followed, blood pressure and blood volume were carefully controlled, and after recovery from the anesthesia, little could be found of a residual weakness. The finding of vascular insufficiency of any major organ should bring one a little nearer surgical therapy if bleeding does not stop.

5. Rate and manner of bleeding.—All recent articles concerned with bleeding peptic ulcer have emphasized the importance of the rate and manner of bleeding after hospitalization and institution of therapy (3, 4). There has been fairly general agreement that continued bleeding from a peptic ulcer at a rate which requires more than 1,500 cc of blood every 24 hours to sustain an adequate blood volume is an indication for surgery. Since ulcer bleeding tends to be intermittent and is not usually a steady hemorrhage, some elasticity in the applications of this rule must be allowed. The repeated massive hematemesis and melena of steady hemorrhage may be too great for one to wait 24 hours before transferring the patient to surgery. The trend of bleeding of intermittent type can usually be foretold from a 48 hour period of observation, and the 1,500 cc rule applied to the averaged 24 hour periods. The younger the patient, the longer it is safe to temporize medically, yet no age group can be considered exempt from bleeding of the degree requiring surgical intervention.

Certain aspects of the manner of bleeding carry prognostic significance. These include, of course, hematemesis or, in the very rare instance that this is lacking, red rather than tarry stools. Recurrent bleeding at intervals of many hours but of large size, exceeding 2,500 cc, is serious. Constant gross oozing, 500–1,000 cc a day for over three days, falls into the same category, as does slower oozing over a seven to 14 day period. If bleeding is to recur, it will be demonstrated in 85% of patients by the third hospital day. During these 72 hours, strict vigilance must be maintained. This period of observation could be undertaken on either medical or surgical wards, but it is only on medical wards that

excellent medical ulcer treatment can be administered during the interim. This also avoids the apprehension of operation produced by admission to a surgical ward, thus enabling some patients to escape the necessity of emergency surgery. The surgical member of the team should be prepared to correct any defects of preoperative observation or management.

The significance of previous bleeding episodes as related to the prognosis has been greatly overemphasized. What is important is the chronicity and virulence of the ulcer and the area of its erosion. I have seen few ulcer patients who had more than two massive hemorrhages. There is a group who have experienced three, five, 15 or 30 minor bleeding episodes without ever needing more than an occasional single transfusion. These patients tend to follow a mild pattern of bleeding and, if operated on, are found to have no visible vessel in their ulcers. The majority of previous bleeders have an irregular pattern of one massive episode between several mild or moderate hemorrhages. Careful study of the major bleeding episode will usually uncover reasons for its severity. Finally, a high percentage of bleeding ulcer patients requiring emergency surgery have never bled before.

CONTINUED BLEEDING WITHOUT KNOWN SOURCE.—It is appropriate to discuss at this point the problem of continued bleeding without a known source. As mentioned, this group comprises at least 10% of all massive bleeders. Less than 10% of these "unknowns" will continue to bleed after the institution of the usual medical therapy. It has been my policy to carry these individuals on the medical program for a longer period than an ulcer patient, but never to deny them the privilege of exploration. If hemorrhage continues at a rate requiring over 3 L of blood a day for 48 hours or 2 L a day for 72 hours—12 transfusions in each instance—further delay without operation cannot be justified. The operation should include exploration of the gastrointestinal tract, to discover the highest level of blood within the tract, and a high subtotal gastric resection if it is apparent that the source is the "upper" tract. Superficial gastric erosion, particularly when associated with submucosal vessels or cirsoid aneurysms, often cannot be found by the usual exploratory techniques. Careful inspection of both gastric and duodenal stumps is indicated if the resected specimen does not contain the bleeding source. Personal experi-

ence influences us all a great deal. The incredible sight of blood spurting from apparently normal mucous membrane in a case of cirsoid aneurysm helped mold my thoughts in this fairly radical direction. Should no source be found at gastrectomy, little harm will have been done by a good surgical team, as the duodenal stump problem will have been minimal with an uninvolved duodenum.

GASTROINTESINAL BLEEDING IN SPECIFIC ANATOMIC AREAS

Bleeding above the esophagus should be readily visible to the examiner who has noted that the patient has spat or coughed out his blood rather than truly vomited it. Such lesions belong in the province of the general surgeon or otolaryngologist and will not be further considered here.

Esophagus

Between 7 and 8% of upper gastrointestinal bleeding has its origin in the esophagus. Almost all massive bleeding from this tube has its source in esophageal varices, but esophagitis with peptic ulceration appears often enough to warrant comment. Tumors, particularly carcinoma and leiomyoma, usually fail to evoke more than mild hemorrhage and are easily recognized by the symptoms. A diverticulum almost never is a focus of bleeding but may be if torsion or other changes produce infection and vascular insufficiency. Aortic aneurysms may rarely erode and rupture into the esophagus, but at such a stage there is little that a physician can do but sedate the patient and set in motion the plans for local disasters. Hemangiomas do occur in this area and may be seen by esophagoscopy.

Esophagitis and esophageal ulcer rarely appear spontaneously, but follow displaced gastric mucosa, trauma—either chemical or mechanical—and particularly hiatus hernia. There seems to be a rising incidence of the last. This may reflect the recent realization that much of the bleeding associated with hiatus hernia comes not from the herniated stomach but from the peptic esophagitis that accompanies the hiatal lesion. With development of the

hernia, the cardia of the gastric esophagus loses its external sphincter—the diaphragm. Regurgitation may then appear. A frequent complication of this is peptic esophagitis. Should this be untreated, it may rapidly develop into a significant ulceration. Both the esophagitis and the ulceration can be easily recognized by the radiologist if he focuses his attention during fluoroscopy on the esophagus above the hernia itself. The presence of reflux should draw his attention to this area. Often the patients give a history of peptic ulcer and have high gastric acidity. Then with age, or perhaps after abdominal trauma, the hiatus hernia develops. Initially it may be asymptomatic. But if the loss of the external sphincter of the cardia permits reflux, burning distress after meals reappears. This time the pain is a little higher, behind the xyphoid, and on careful questioning one may elicit a history of some type of dysphagia, usually the first few swallows or of certain foods. If untreated, the symptoms may increase. Hemorrhage can be massive and is then invariably associated with hematemesis of bright red blood.

The x-ray picture is characteristic. If any doubt regarding the presence of cancer exists, esophagoscopy should be performed and a single biopsy specimen taken. Repeated biopsies may lead to more inflammation and the hazard of stricture. Should stricture already be present, appropriate bouginage is indicated after the esophagoscopy.

Medical treatment at a time of mild or moderate bleeding includes the standard therapy. (1) For the hiatus hernia—small, dry meals with liquids between meals; avoidance of increased abdominal pressure after eating; not lying down for two hours after a meal. (2) To reduce gastric acidity—bland diet; antacids such as Gelusil, 10 cc three-quarters of an hour after each feeding and at bedtime; antispasmodics and sedatives. It is not clear whether an effective parasympathetic depressant such as Banthine does more good by decreasing gastric secretion than harm by excessive depression of gastric motility and prolongation of emptying time. This may result in a greater likelihood of regurgitation and reflux into the esophagus. Treatment of each patient must be individualized. (3) For the esophagitis itself—soft, nonirritating foods, well chewed before swallowing. Often pain can be avoided by a small feeding of a warm bland liquid at the begin-

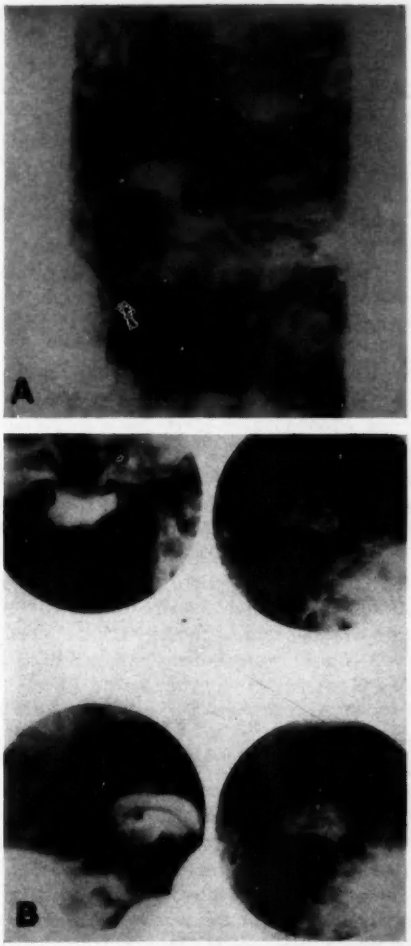

FIG. 1.—*A*, demonstration of a large gastric ulcer at the angulus of the lesser curvature of the stomach with a small amount of barium. *B*, spot films taken in various positions indicate size and depth of this ulcer.

ning of each meal. At the end of each meal and whenever any burning substernal pain appears, the patient takes 5 cc of a prescription prepared by adding just enough olive oil to a mixture of calcium carbonate and magnesium oxide powders to make a sticky paste. This coats the area of esophagitis better than Gelusil The calcium carbonate and magnesium oxide are usually mixed in a 10:1 proportion.

Treatment by this program should be followed by rapid loss of symptoms within a few days and complete loss of the esophagitis, as demonstrated by x-ray, within a month. Since the fundamental situation that produced the esophagitis still exists, therapy against gastric distention and reflux must be continued. A similar program will be effective against esophagitis in the absence of hiatal hernia.

When massive bleeding occurs, oral feedings should be discontinued, the patient kept in bed with 12 in. bed blocks at the head end, appropriate sedatives and antispasmodics given, and hourly medication with alternate Gelusil and calcium carbonate paste administered. Blood lost should be replaced by transfusion. The passage of a soft gastric tube into the stomach to place that organ on constant suction is more apt to irritate the area of esophagitis than help by keeping the stomach empty. When these patients have recovered from the bleeding episode, they should, if under 50, be considered for elective repair of the defect of the diaphragm. A long trial of medical therapy can be more safely given those in the older age groups, since age tends to reduce both gastric acidity and tonus.

Esophageal Varices

Of all the causes of massive bleeding, management of the bleeding esophageal varix secondary to cirrhosis of the liver leaves most to be desired. This is due not so much to the fact that the bleeding cannot be controlled or lessened by medical or surgical means but to the disastrous effect that massive or repeated bleeding has on the liver itself. For this reason the incidence of disaster is least in the patient with extra-hepatic portal block; second in the individual with healed hepatic necrosis such as follows infectious hepatitis; third in the patient with Laennec's cirrhosis who

has not been drinking and has been eating; and most discouraging in the cirrhotic who has been drinking and has had his current spree halted by hematemesis and hospitalization. Throughout the country, hospitals which care for the usual proportions of these four types report the same sad immediate mortality statistics—approximately 70% when massive bleeding has taken place. After a slight bleeding episode 50% will die within the year. No disease other than cancer has a worse record. It is unfortunate that the largest group includes those we can help the least, the chronic alcoholics. Certain advances in our knowledge of the metabolically deranged liver give hope for better results in the future.

The experienced clinician has little trouble spotting an alcoholic cirrhotic, but the postnecrotic cirrhotic often lacks all the stigmas—florid face, spider angiomas, ascites, large liver and spleen. The spleen may become palpable after blood replacement, and its size should always be rechecked. The history may be most important, particularly a story of some months or a year of loss of sense of well-being in the past, perhaps while the patient was in an endemic area of hepatitis. As a rule, these patients have had no clearcut history of jaundice. Most of my experience with this group has been with former service men who had acquired their liver infection in the European, Mediterranean or Pacific theaters. Many patients have developed the same disorder in this country. There is also a group of young women who have acquired subacute or chronic hepatitis of the type most apt to go unrecognized and consequently remain untreated. As a rule, the postnecrotic cirrhotics have a considerable degree of portal hypertension and some splenomegaly. Their varices are large and easily recognized by x-ray with Rugar barium, even during bleeding. Endoscopy is helpful, but a high degree of experience is necessary to perform it during bleeding, and there are greater hazards to its performance by inexperienced physicians than there are to x-ray study.

The typical patient with Laennec's cirrhosis usually has moderate-sized varices, that can be visualized by x-rays in about 65% of these patients. Because this group has a high incidence of hiatus hernia, gastritis and peptic ulcer, extremely careful sifting of the recent history is necessary to obtain clues pointing to the

actual source of bleeding. The reassurance that an emergency G–I series gives in these circumstances cannot be underrated. Esophagoscopy, if performed at the time of active hemorrhage, will disclose the bleeding varix. The emergency bromsulfalein test is only indirectly helpful—normal value during bleeding is not in keeping with the diagnosis of cirrhosis.

Certain broad principles of management of bleeding varices can be laid down. It is not generally recognized that about 60% of esophageal varices have some ulceration as the primary cause of fatal hemorrhage. In 40%, spontaneous rupture takes place due to the increased hydrostatic pressure in the varix. Palmer (5) has clearly demonstrated the significant rise in portal pressure that occurs during the Valsalva maneuver or to a lesser degree after physical effort. Varices are essentially at the same level as the right auricle, so posture should have little effect on their hydrostatic pressure. Yet radiologists have for years noted that varices are demonstrated much more easily with the patient in the horizontal than in the erect position and that as the patient is tilted from horizontal to erect the varices become less readily demonstrable. For this reason and the high incidence of ulceration presumably from reflux or regurgitation, it is our policy to maintain a head-up position of approximately 15 degrees if the circulatory status of the patient permits. Bed blocks under the head of the bed and a good footboard are necessary for this purpose.

In the past it was considered good practice to treat these people as ulcer patients, since neutralization of the gastric contents prevents further ulceration of the esophagus. Should reflux take place, digestion of the recent thrombus in the varix may occur. If the episode of bleeding has been slight, manifested only by brief melena, this program of neutralization, small feedings, proper positioning and antispasmodics should be continued. Fairly good results may be expected on this program unless the hepatic disorder is severe or associated with a disorder of the clotting mechanism.

More difficult decisions arise when bleeding is more massive. After many changes in our policy, the experience gained with over 40 patients with bleeding varices has finally crystallized our management. In patients whom we have previously studied and

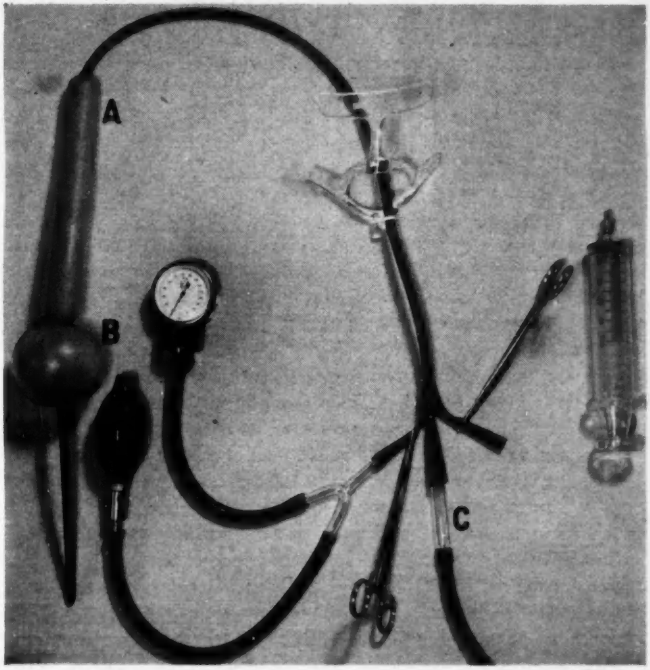

FIG. 2.—Equipment used in operation of the Sengstaken double balloon tube. *A*, esophageal balloon, 20–35 mm. Hg; *B*, gastric balloon, 200–250 cc.; *C*, gastric suction tube. Plastic face anchor fits around the nose with pressure distributed on forehead and cheeks by the broad plastic. In practice, the lower prongs are padded if the tube is to remain in place over 24 hours. Here, the tube is attached to the anchor by a thin strip of adhesive tape, but more tape is used in actual fixation. The anchor knob is so placed that it is adaptable for a tube in either nostril.

know have large esophageal varices, the Sengstaken tube is promptly passed and the balloons inflated. As soon as bleeding is well controlled and the patient restored to his maximal circulatory state, he is transferred to the operating room. There, through a transthoracic approach, with the lower esophagus opened for a distance of 4 cm, the varices are ligated by multiple over-and-over obliterative sutures, beginning in the cardia of the stomach and working up the esophagus as far as the incision permits. Supportive measures for the liver are instituted postoperatively, and as soon as the patient's condition permits, a portacaval shunt or other anastomotic procedure is done. It is not wise to delay the second operation beyond three weeks, as the ligation procedure is purely a temporary expedient. It has been successful, however, in halting hemorrhage from large varices for a month. The individual without a previous diagnosis and whose emergency G–I series discloses large varices is also treated in this fashion.

Patients known to have or found to have small or modest varices are watched after admission on a conservative program. Upon *any* conclusive evidence that fresh bleeding has occurred, they are prepared for insertion of the Sengstaken tube and this procedure performed. Intelligent special nursing is most helpful.

SENGSTAKEN TUBE (Fig. 2).—It is appropriate at this time to recount certain techniques and policies in the use of this tube, because it is an effective procedure only when the tamponade is carried out accurately and carefully. First, the balloons and tubes should be checked for leaks and patency. The patients, being alcoholics, do not take the passage of tubes with ease and simplicity. Therefore, application of a minimal amount of local anesthetic to nose and back of throat will reduce considerably the discomfort, straining and retching attendant on the passage of the tube. The pharynx must not be completely anesthetized because the accumulated secretions in it will not be noted and may be aspirated into the tracheobronchial tree. Next to proper placement of the gastric balloon, the hazard of aspiration is the greatest complication of the use of the double balloon tube. The gastric balloon is the keystone of the whole system. It should be fully inflated on passage of the tube beyond the 50 cm marker, then pulled back until it catches at the esophageal cardia. It is then carefully taped to an appropriate anchor. The 150–200 cc

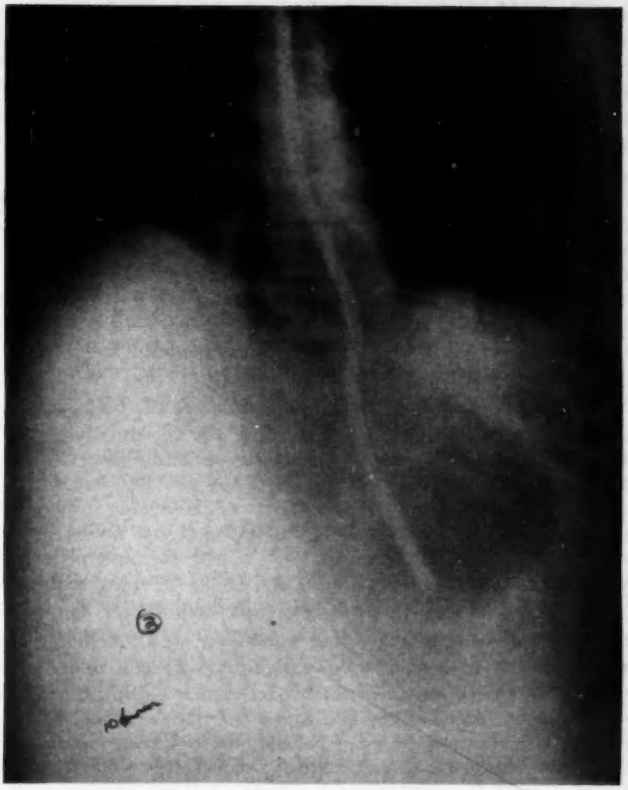

FIG. 3.—Failure of gastric balloon to be properly positioned due to hiatus hernia in stockily built, chronic alcoholic, age 48, with recurrent massive hematemesis. Bleeding continued after placement of tube, and routine over-penetrated portable x-ray film shows the balloon in the position most often found in hiatus hernia. After addition of more air to the balloon, bleeding ceased. High position of the esophageal balloon created a problem in aspiration of secretions, and he died of hepatic insufficiency and multiple aspiration pneumonia. In retrospect, obliteration of the upper part of the esophageal balloon, or tracheotomy, might have prevented death. Autopsy confirmed the presence of a sliding hiatus hernia.

of air first recommended has proved inadequate, and 275–300 cc has been more successful. Suction on the stomach tube is begun. The esophageal balloon is then inflated *slowly* to 20–25 mm Hg of pressure. A few extrasystoles may appear during this period. The stomach tube is then irrigated with water to prevent clogging from blood clotting, and this should be frequently repeated. The pressure in the esophageal balloon will drop as the esophagus relaxes, and frequent additions of air to this balloon are necessary. The pressure often required to stop bleeding completely is 30–35 mm Hg. This will need frequent checking by the doctor and nursing attendant.

By this time improvement in the color of the gastric aspirate from red to black should be noted. It is most essential to check now the position of the gastric balloon by a slightly overexposed (portable) x-ray film of the upper abdomen and lower chest. Failure to note improvement in the character of the aspirate is an especial indication for determining if the keystone is in position. Figure 3 shows an example of the difficulties occasionally encountered—a sizable hiatus hernia. Other problems include too low placement of the gastric balloon or inadequate anchoring, with gastric contractions pulling the whole device several centimeters down the gastrointestinal tract. The strength of gastric contraction and peristalsis is amazing. If the bleeding is coming from esophageal varices, we believe that hemorrhage should be promptly halted by properly placed balloons. Continued bleeding is an absolute indication to check positioning and to look for mechanical interference such as hiatus hernia. Failing these, one must consider another possible source for the bleeding. In two instances we have introduced barium through the gastric tube and found peptic ulcers, which then successfully received appropriate surgical therapy.

Adequate anchoring is a problem, and after many unsuccessful and crude attempts which usually ended with some necrosis of the alae nasi, we were fortunate to have the assistance of a skilled dentist interested in plastics. Donald F. Gearhart, D.D.S., of the Boston Veterans Administration Hospital, perfected the plastic face piece (Fig. 2) with a good anchoring section adaptable to either nostril. Using this device, we have kept tubes in place and in proper position for over a week without great dis-

comfort to patient and without tissue injury from pressure of the tube.

A third factor of importance is adequate aspiration of the posterior nasal and oropharyngeal secretions evoked by presence of the tube. These must be looked for and constantly removed by suction, or aspiration pneumonia will take place. In drowsy or comatose patients we put down a nasal catheter for this purpose and, failing this, consider elective tracheotomy. Nursing attention to this matter must be constant and vigilant.

It is important to begin feeding these patients bland, caloric liquids in small amounts through the gastric tube as soon as the aspirate has become clear or of only slightly coffee-ground appearance for eight hours. Frequent checking on retention of these feedings is necessary at the start. Finally, decompression of the balloons must be carried out in an orderly sequence after 48–72 hours of effective control. The esophageal balloon pressures are reduced 4–6 mm Hg every two hours beginning the third day, until decompression is complete. The fourth day, gradual deflation of the gastric balloon is effected in 50 cc amounts. After complete deflation, it is left in place a few hours and vigorous suction applied to the gastric tube during this period. If no fresh red blood is noted, the entire system may be gently withdrawn. If bleeding occurs, both of the tubes should be reinflated for 48 hours, then the process of deflation repeated. It is amazing how well the esophageal mucous membrane tolerates the tubes, even for many days. We believe it best to continue therapy with the tube after one failure, rather than switch to surgical ligation.

Hepatic insufficiency.—The next and perhaps greatest problem in the care of these patients is the prevention of acute hepatic insufficiency or, if it has already developed, the treatment of this serious complication. There are few more discouraging situations in medicine today. Some progress has been made, and we can be hopeful. Prophylaxis is always better than cure of the fully developed disorder, so everything should be done to aid and spare the liver from the start. In general, the patient who arrives at the hospital in a metabolically poor nutritional state from recent active alcoholism is the one most apt to get into serious trouble. His liver is usually quite large, but may be small, and is always fatty. The cell nuclei are squeezed into the rim of fat that has

replaced the normal cytoplasm. Alcoholic hyaline may be visible in the cytoplasm elsewhere.

This patient must be fed as soon as possible. This is done through the gastric section of the Sengstaken tube. It is desirable to include in the 24 hour total, 2,000 calories and not over 60 Gm of protein. With skimmed milk as a base, glucose, eggs and protein hydrolysate can be mixed for this purpose, with water added to dilute to 2,800 cc. The patient should be turned slightly to his right to assist gastric emptying and place the cardia of the stomach as far from gastric fluid as possible. Before each new feeding, suction should be applied, and if the stomach is not emptying adequately, the volume of each feeding is reduced. It is essential to prevent increased gastric distention. If frequent or soft stools appear, the formula should be immediately changed to skimmed milk, with 2 eggs in each liter, omitting all glucose and protein hydrolysate temporarily. Glucose, 1,500 cc of 15% solution to which appropriate vitamin B complex and vitamin C preparations have been added, can be dripped through a small polyethylene catheter in a vein at a rate of 5–15 drops per minute. The intravenous glucose therapy should be started early in hospitalization, as soon as transfusions have replaced the blood lost by hemorrhage, and be continued during the tube feedings until the hazardous first 96 hours are past. Crude liver extract, 3 cc daily intramuscularly, is alleged to favor hepatic repair. It can do no harm. If tolerated, oxygen should be given by face mask, as any reduction in hepatic anoxia, theoretically at least, should be most beneficial. There are good animal experiments supporting this theory. Another possible aid is certain antibiotics, based on the evidence from Reyneirs' "Germ-Free Life Laboratory" where such medication prevented death or liver necrosis in animals. In rats fed a necrogenic yeast diet, the appearance of hepatic necrosis was markedly delayed or prevented by Aureomycin. Severe bleeding in an active alcoholic warrants the institution of Aureomycin intravenously, 1.0 Gm a day for four days.

When hepatic coma or acute hepatic insufficiency appears incipient, more radical measures become necessary. Because of the manifold functions of the liver, the insufficiency may manifest itself clinically in many ways. Some patients may have the flapping tremor of their hands, at the wrists; others become drowsy

and some febrile. The English have commented that the alcoholic who previously had been a surly, rude individual and then becomes placid and cheerful is in impending coma. This reaction does not seem to occur frequently among the owners of damaged American livers. Most often, one can detect a subtle but clear cut change in "awareness of consciousness." This is easily manifest if any simple intellectual test is given the patient. The subtraction of serial seven's from 100 may be done fairly well the first day, but instead of improving, answers become less intelligent on the second day. By the third day one may be greeted with obscenity instead of accuracy. Restlessness that night leads to the administration of paraldehyde, and on the fourth morning coma is in full bloom. Chlorpromazine, which works so well in reducing agitation from various causes, may produce jaundice, but not from a hepatoxic effect. It must be used in one-half the usual dose, 10 mg intramuscularly, and, as with a narcotic, each dose must be ordered separately after the patient has been observed. There is no safe sedative, and chlorpromazine seems to be the drug of choice, if one must be used.

There are physicians who have maintained their fourth sense—smell—as an aid in diagnosis. This atavistic trend toward our canine friends or the medical teachings of the late 19th Century should be encouraged, as the ability to recognize fetor hepaticus will be of great help in picking out the patient whose liver is doing badly. Although it is not necessarily a bad prognostic sign in infectious hepatitis, its appearance in a cirrhotic with massive bleeding has universally carried an ominous connotation in my experience. Butt (6), who has written a stimulating review of this subject, describes the odor as somewhat sharp and pungent or a scorched fruity odor near the mouth, but a more sweet and not unpleasant smell at a distance. He points out that a similar odor can be obtained in the urine, and isolated a tertiary amine similar to 2-methylpiperidine from urines of patients having the fetor.

Acute hepatic insufficiency is usually associated with a high level of blood ammonia, and coma may indeed be induced by medicine containing ammonium or by a very high protein diet. Relatively few hospital laboratories are prepared to test for blood ammonia. A rising serum bilirubin content, falling serum cholesterol-cholesterol ester ratio and a very low blood urea nitrogen

level are time-honored laboratory tests of impending hepatic coma.

Treatment of hepatic coma itself, other than removing the major precipitating cause and correcting any obvious metabolic or electrolyte abnormalities, has left much to be desired. Intravenous glucose and tube feedings have already been instituted in our patient. Aureomycin, if not already in use, should be administered in 1.0 Gm doses intravenously each day for four to five days. Besides the hazard of staphylococcic enteritis, this drug may in itself produce histologic changes in the liver if given over longer periods. These fatty changes in the liver cell are different from those found in alcoholic degeneration. In recent years the British have become somewhat enthusiastic over the intravenous administration of glutamic acid, based on the theory that this substance combines with ammonia to form glutamine, thus removing ammonia from the circulation. Sodium glutamate has been used in this country, in my brief experience, with very doubtful benefit. Others have been more enthusiastic. Some have obtained the sodium glutamate by using the household meat flavorer "Accent," 23 Gm of which is added to 90 cc of water, autoclaved and administered in 500 cc of 5% glucose in distilled water over a four hour period.

A more abundant experience with large intramuscular doses of cortisone, 800–1,000 mg a day, has led me to favor this drug. It will certainly reawaken the comatose patient and start him eating again. The effect may not be lasting, particularly if bleeding recurs. It is important to remember the effects of cortisone on gastric secretion and accordingly neutralize the rising acidity by frequent feedings, antacids or aspiration if the Sengstaken tube is still in place. If this is done meticulously, an appreciable number of survivals may be obtained.

Stomach

Hemorrhage from gastroesophageal lacerations at the cardiac orifice of the stomach—Mallory-Weiss syndrome—is a rare cause of massive bleeding. It is often unrecognized and fatal. It may be found in 1–2% of autopsies on patients dying of uncontrolled bleeding. A history of violent or protracted retching and vomiting

before the onset of bleeding may be obtained. It occurs in alcoholics or in an older, nonalcoholic age group with atrophic gastritis. Hemorrhage may be very severe and rapid. Diagnosis by x-ray or endoscopy is difficult because of the nature of the laceration and the excessive bleeding. The lesion can be seen, however, if the proximal stump of the high gastric resection is examined at emergency operation. Should the lesion be suspected on clinical grounds, a program of no oral feedings or fluids, sedation and parenteral administration of adequate doses of atropine and chlorpromazine should be tried. Further vomiting must be avoided, as fresh bleeding occurs with each paroxysm.

Hiatus Hernia

Bleeding in this condition may be from peptic esophagitis, gastritis within the herniated portion of the stomach or a gastric ulcer. The usual site of the ulcer is the neck of the sac where it passes through the diaphragm. Massive bleeding may occur in this last instance. The age, build of the patient and postural symptoms will help us suspect this condition. It is easily found on the emergency G–I series. If a gastric ulcer is present, subtotal resection is in order, but the less serious sources associated with herniation should be managed by the customary small feedings, upright posture and antacids just described.

Gastric Ulcer

There is no need to restate the management of the patient bleeding from a gastric ulcer, as it has been discussed in detail under general management. It is a most serious matter, and any significant recurrent bleeding after institution of therapy is an indication for surgery. Medical treatment must be especially meticulous to avoid recurrence and should include cessation of smoking, avoidance of all emotional or gastric irritants and an exceptionally careful feeding and antacid program. In borderline cases, the fear of malignant ulcer may be introduced as an indication for operation. A little-stressed but highly significant factor in weighing this matter is the family history of carcinoma, particularly of the gastrointestinal tract. When present, the likelihood

of the ulcers being malignant is increased from 8% to 25%, in my experience; therefore a positive family history becomes a factor of importance in the management of the bleeding episode.

Gastritis

Gastritis appears to be an increasingly frequent cause of both mild and massive gastrointestinal bleeding. Its incidence varies from 10 to 20%, depending on the age and type of patient population involved in any study. Factors responsible for the rising incidence seem to be largely related to alcohol, aspirin and anxiety, either singly or in combination. The history for each requires a recent exposure to that particular irritant. The alcoholic is on a spree and vomits first coffee-ground material and then red blood. X-rays will fail to show varices or ulcer, and subsequent gastroscopic examination may reveal the cobblestone appearance of hypertrophic gastritis with areas of erosions or multiple submucosal hemorrhages. Occasionally, the bleeding episode may frighten the alcoholic patient and be used successfully as a tool to separate him from his dependence on alcohol. Under these conditions no attempt should be made to minimize the bleeding; in fact, overemphasis is desirable. Side remarks between physicians at gastroscopy are particularly apt to leave an impression. It is exceedingly unusual for the bleeding from alcoholic gastritis to continue more than three days after withdrawal of alcohol.

A different and more serious group of bleeders includes individuals who require frequent or daily use of salicylates. A few are individuals with recurrent headaches, but the greatest number are older or elderly patients, most often females who take 6–10 aspirin tablets, or the various combinations of salicylates, daily for relief of the aches and pains of the aging processes. They may do this for years, then apparently develop a local sensitivity reaction in the stomach associated with hemorrhagic gastritis. The first bleeding episode may be minimal, but further use of salicylates may cause exsanguinating bleeding necessitating surgery. It is important for the practitioner to remember that although some patients can safely switch to enteric-coated aspirin or Bufferin, there are others who will bleed again on exposure to these drugs. Complete abstinence from all varieties and types of salicylates is

essential, but difficult when advanced osteoarthritis is harassing the patient.

There appears to be a higher incidence of gastritis and peptic ulcer when cortisone and salicylates are used in conjunction in treatment of arthritis. The presence of any heartburn or other gastric distress calls for the adoption of a bland diet program with antacids and antispasmodics and a change in the basic medication for the arthritis. Appropriate x-rays should be taken. The gastritis associated with uremia or other terminal disorders has no satisfactory treatment.

Gastric Tumors

As a rule, tumors of the stomach, whether benign or malignant, do not bleed with great vigor at the start, giving the physician adequate warning to make the indicated gastrointestinal studies and x-rays. These should be done promptly. The finding of hypochromic anemia in any patient over age 30 demands this immediate investigation. As the lesion grows, chronic oozing or intermittent massive bleeding does occur in about 0.5% of all such bleeders. It is difficult from the history or physical examination to suspect tumor, rather than ulcer, whether polyp, cancer, leiomyoma or lymphoma be the lesion in question. X-ray and gastroscopy are the stand-bys for diagnosis. Barring major contraindications, surgical removal at an early date is indicated for all tumors. Even lymphosarcoma should be explored, if nodes are not available for biopsy elsewhere, as it may very rarely be an isolated lesion. If nodes are present, x-ray examination should be first choice. Every patient in whom there is any doubt as to the source of bleeding should have a repeat study about three weeks after the initial investigation. This will pick up the occasional tumor missed during the first examination.

Miscellaneous Gastric Lesions

There are numerous odd causes of bleeding from the stomach not mentioned in the previous paragraphs. Diverticula of the stomach can be a source of fairly severe bleeding on occasion, particularly if peptic ulceration has occurred. The telangiectasias

of hereditary Osler-Weber-Rendu disease may produce bleeding anywhere in the gastrointestinal tract. Their presence can be suspected from the family history of bleeding or the presence of the typical telangiectasias on lips or beneath the finger-nails. Surgery is not apt to be helpful, and treatment should consist of transfusions, bland feedings, rutin, snake venom injections or topical application of thrombin. Various inflammatory lesions of the stomach, rarely seen today, including tuberculosis and syphilis, have been reported to bleed. Gastric granuloma either of sarcoid type or with eosinophilia has also been noted as a source of hemorrhage.

Prolapse of the gastric mucosa through the pylorus is not rare, but a dispute exists whether it alone can be a source of bleeding. I believe the weight of evidence favors it as a source. Treatment should be a conservative bland feeding program with an attempt to paralyze the gastric motility by large doses of atropine or Banthine, for there is some evidence that incarceration of the redundant mucosa occurs in a hypertonic stomach during the peak of antral systole.

Duodenum

Duodenal ulcer is the commonest source of massive bleeding. I have already detailed our program of management and discussed the indications for surgical intervention. Table 3 summarizes these indications. It must always be kept in mind that gastrectomy is hazardous in cases of duodenal ulcer because of the difficulty in adequately turning in the duodenal stump. Since the operation cannot be prolonged, the surgeon must consider means of operation that will stop bleeding. Should an extensive inflammatory mass or a postbulbar ulcer prevent easy turn-in, a two-stage exclusion operation must be done rather than risk a more prolonged procedure. This includes transfixing and ligating the bleeding gastroduodenal artery and its two terminated branches, but leaving the ulcer in situ. Healing usually occurs within a few weeks, but if bleeding recurs further surgery can be done with the surgeon's attention focused on this objective since the anastomosis is already complete. Billroth I operations must not be attempted. There are various means of coping with the

technical problems associated with the duodenal stump. Meticulous placing of the nasal gastric tube well up the afferent loop of the anastomosis is necessary. Equally careful maintenance of its patency with constant decompression of the duodenum is essential. It should not be withdrawn before peristalsis is present and the character and volume of drainage indicate that this is safe—

TABLE 3.—Indications for Emergency Operation for Bleeding Duodenal Ulcers

Indication	Manifestations	Significance and Action
Rate of bleeding	1,500 cc blood required every 24 hr for 2 days to maintain adequate blood volume, plus repeated hematemesis; prolonged oozing 500–1,000 cc for 4 days	Recurrent arterial bleeding or large venous flow demand surgery Surgery indicated
Manner of bleeding	Sudden massive episode necessitating 2,500 cc blood to bring out of shock with hematemesis	Large arterial bleeding demands surgery
Source of bleeding	Recurrent episode of bleeding where source is postbulbar ulcer	Arterial bleeding with or without pancreatic erosion; surgery if recurrent bleeding
Associated complications	Cirrhosis of liver; chronic nephritis not in uremia; detectable insufficiency of any major organ during period of hypotension	Surgery if significant recurrence of bleeding after blood replacement and institution of therapy

about three days. A second tube often must be placed in the gastric cuff for 24–36 hours to clear it of secretion or clots. Attention to these details should greatly reduce blow-outs of the duodenum.

Massive hemorrhage has been reported from duodenal diverticula, tumors of the duodenum, ampulla of Vater, bile ducts and from pancreatic erosions secondary to tumor or pancreatitis. Congenital anomalies, hemangiomatous lesions and other bizarre pathologic disorders may occur in the first foot of small bowel. When bleeding occurs from one of these, in the absence of hema-

temesis or localization by emergency G–I x-ray study estimation of the approximate site of origin should be attempted. The Einhorn string test or the passage of a long, soft gastric tube with the patient on his right side is easily and safely done. Should the tube reach the ligament of Treitz without finding blood, it should be withdrawn and a Harris or Miller-Abbott tube inserted in its place for more distal small intestinal study. Fortunately, it is most rare to have rapid bleeding from these areas. Surgical exploration is necessary if it does take place.

Small Intestine

By far the commonest cause of small bowel bleeding is man-made, namely, marginal or jejunal ulcer following gastrojejunostomy. Although it occurred in only 1% of our series, most reports suggest a greater incidence. In 97% of all patients who have had a gastrojejunostomy, recurrent bleeding will arise either in such an ulcer or from gastritis of the remaining stump. Pain, if present, suggests the presence of ulcer. Its location often is atypical—left or right, mid- to lower quadrants. The ulcer may be 10 cm or more from the anastomosis. Jejunal ulcers are notoriously difficult to spot by x-ray study, 45% success being about the average reported. Occasionally gastroscopy helps to establish the diagnosis. Most are small ulcerations and do not bleed massively, but large ulcers can occur and produce exsanguination. Treatment of the gastrectomized patient is conservative medical. In the patient with a posterior gastroenterostomy and a demonstrable marginal ulcer niche, the same indications for surgery exist as for a duodenal ulcer. A nonfunctioning posterior gastroenterostomy and hemorrhage suggest that bleeding is occurring from the original duodenal ulcer.

Less common, but more interesting, is the study of bleeding from certain other lesions, particularly small intestinal tumors. Their hemorrhage usually is a slow ooze, with the development of anemia, but they often produce tarry stools or, if the bleeding is massive, dark red blood by rectum. The presenting symptoms are bleeding and poorly localized abdominal pain. Diarrhea suggests an inflammatory lesion such as regional ileitis, but it can occur with lymphoma. Primary carcinoma, intestinal

lymphoma and benign tumors occur with about equal frequency.

In at least two conditions, careful inspection of the patient will suggest important leads in diagnosis. These are the melanin spots of the oral mucosa, lips and digits associated with intestinal polyposis (Peutz-Jeghers syndrome) and the café-au-lait patches or subcutaneous neurofibromas associated with gastrointestinal neurofibromatosis. Occasionally, a tumor may be felt on palpation. It will fall on the radiologist, in most instances, to establish the diagnosis.

Several x-ray techniques are available. In order of simplicity of execution they are: (1) suspensions of 4 oz (120 cc) of barium in 5 oz (150 cc) of isotonic saline solution taken by mouth, with a succession of films of the abdomen every half-hour; (2) administration of the small intestine enema through a nasal tube passed into the proximal jejunum, with the entire small intestine then filled with barium; (3) retrograde study by barium enema if the ileocecal valve is incompetent; (4) passage of a Miller-Abbott tube to the point where guaiac-positive aspirate is obtained, then injection of barium through the tube and the taking of appropriate spot films.

Of the benign tumors, hemangioma, leiomyoma, adenoma and neurofibroma are most apt to produce massive bleeding. All of the malignant tumors can bleed briskly, carcinoids (argentaffinomas) and lymphomas particularly, as these lesions are frequently multiple. The bleeding tendencies of primary adenocarcinoma of the small bowel help in its early detection. Metastatic tumors may also hemorrhage.

INFLAMMATORY LESIONS.—Regional enteritis is a common disorder, and approximately 20% of patients have significant bleeding during the course of the disease. It is rarely massive. The other symptoms, diarrhea, colic and weight loss, suggest the presence of this disorder. The diagnosis is readily established by x-ray examination. Tuberculous enteritis, intestinal sarcoid and intestinal lipodystrophy (Whipple's disease) have all been reported as causing melena. They are rare and do not bleed massively.

MISCELLANEOUS.—Anomalies of the small intestine are not rare and may be associated with alarming bleeding. Meckel's diverticulum with symptoms is most common in boys under 15,

particularly infants. Other diverticula, particularly in the jejunum, may be a source of severe hemorrhage. Intestinal duplication in its various forms has been reported as producing hemorrhage, particularly if the terminal ileum is involved.

Primary ulcers of jejunum or ileum do occur rarely, usually opposite the mesenteric border of the intestine. The symptoms may mimic peptic ulcers elsewhere. Vascular disorders, such as periarteritis nodosa, purpura and blood dyscrasias, are intrinsic causes of melena and may be recognized as part of the primary disease. Henoch-Schönlein purpura is the most common of these. Azotemia in chronic nephritis may cause exsanguinating hemorrhage. Pylethrombosis, foreign bodies and rarely hypertension may be associated with intestinal apoplexy. Intussusception in children does not require an associated lesion for its production. In addition to the colicky pains and presence of a mass, blood is often passed by rectum. Bleeding is rare in the adult type. Mesenteric thrombosis or any other severe abdominal catastrophe associated with hemorrhagic infarction of the intestine may be associated with bleeding.

Colon and Rectum

Cancer and polyps.—Cancer of the colon should be considered the commonest cause of colonic bleeding, overt or occult. It rarely causes massive exsanguinating hemorrhage, this usually being secondary to ulcerative colitis, diverticulitis or internal hemorrhoids. Inasmuch as tumors, benign or malignant, call for the greatest care in diagnosis and treatment, they will be discussed first. Mucosal polyps are usually adenomatous tumors of sessile or pedunculated nature. The papillary type of adenoma, the so-called villous tumor, is worth differentiating because of its tendency to recur after local removal. Another group of interest is the congenital or familial type of multiple polyposis with its almost inevitable tendency to become malignant. Approximately 40% of patients with polyps have rectal bleeding as the complaint which sends them to their physician. Since at least one third of these patients also have hemorrhoids or a fissure as an apparent source of the blood, it must be repeatedly emphasized that hemorrhoids can be accepted as the cause of the bleeding only after

digital examination, proctoscopy and barium enema have ruled out other sources for the hemorrhage. Polyps are not a source of serious blood loss, but their detection and proper treatment are most essential.

Almost no cancer of the internal organs has as good a statistical prognosis as carcinoma of the colon and rectum. Five year cures of 60% are commonly reported. Every physician is therefore justified and obliged to exert especial effort and care in his study of colonic bleeding. Bleeding is almost never massive—occult from the right colon, overt from the left colon and rectum. Almost all patients with rectal cancer have large hemorrhoids as an apparent source of blood. Digital examination is the minimum examination of the patient who has the hemorrhoidal type of bleeding—blood following the act of defecation. If blood is on the stool and increased mucus is present, a complete lower bowel study with proctoscopy and barium enema is necessary. Up to 50% of rectal and left colon lesions manifest their presence by blood in the stool. Constipation is a more frequently noted symptom. The two complaints appearing together require a complete investigation in all patients, male or female, over the age of 25. Colonic cancer has appeared in a younger age group, in my experience, than the other gastrointestinal malignancies.

Other tumors can produce bleeding. Carcinoids of the colon or rectum appear to be more malignant than those arising in the appendix. Lymphosarcomas, other lymphomas, neurofibromas and angiomas very rarely produce large bowel bleeding.

Ulcerative colitis.—This may be associated with colonic hemorrhage in three ways: (1) by the acute pathologic process itself involving local or widespread destruction of the mucosa and submucosal structures associated with the disease; (2) after the use of broad spectrum antibiotics, cortisone or cathartics during the course of the disorder; (3) related to the development of polyps or cancer in chronic ulcerative colitis. Bleeding secondary to the colitis carries the most serious immediate hazard, as it may be massive. It is most often associated with an acute fulminating stage of the disease. If hemorrhage is recurrent and massive, ileostomy and colectomy, if possible in separate stages, may be life-saving. Prevention of hemorrhage is important. The use of antibiotics for colitis has been followed by the appearance of staphylo-

coccic infections of the colon more severe than the ulcerative colitis itself. The broad spectrum antibiotics in particular should not be used routinely in its treatment. Giant ulceration of the bowel with exsanguinating hemorrhage has appeared during treatment with cortisone and corticotropin. Whether the antibiotics given in conjunction with this therapy have had a bearing on this, I do not know, but Kirsner and Palmer (7) have seen little of this complication, and they use antibiotics sparingly. Polyposis associated with chronic ulcerative colitis appears usually after several exacerbations and remissions. The increased incidence of carcinoma in ulcerative colitis can also be traced to chronicity. I do not believe that cancer is a hazard until 10 years of disease have effected their changes on the colonic mucosa. Between 10 and 20 years, the incidence of cancer may rise to 10%, an increase of twentyfold. It is often highly malignant, with bleeding or obstruction. The appearance of gross bleeding in a patient with chronic colitis should be a source of concern to the physician and a stimulus for a complete resurvey of the rectum and colon. Prevention by total colectomy is the only logical treatment of extensive polyposis resulting from 10 or more years of chronic colitis.

Other infections, foreign bodies, anatomic, mechanical and vascular disorders of the lower bowel may produce bleeding. Only two require special comment—diverticulitis and diverticulosis, and hemorrhoids.

Diverticular disease.—Diverticular disease of the colon is not as rare a cause of bleeding as has been reported in the past. Fifteen per cent of the patients will bleed, the blood loss being occult, melena, bright red or massive. The last requires prompt action, as exsanguination can occur. The patient is usually past 60 and hypertensive. Previous low, crampy abdominal pain is followed by passage of red blood and clots. Prompt digital examination and proctoscopy will further convince the physician of the low origin of bleeding. Emergency barium enema will show diverticulitis of the colon, and passage and suction of a soft tube into the stomach fails to aspirate coffee-ground material. A G–I x-ray series should also be considered before operation. Continued bleeding of 1,500 cc or more a day is an indication for early operation with laparotomy, colostomy and resection of the sig-

moid. On occasion, the x-ray picture is that of only diverticulosis, but in all patients in my experience on whom operation has been performed, the pathologic finding was diverticulitis. The bleeding points may be difficult for the pathologist to find.

It is of the utmost importance for the clinician to remember that diverticulitis and carcinoma of the colon may both be present. Of great assistance in this differentiation is the appearance of x-ray improvement after 10–14 days of conservative therapy, when diverticulitis alone is present.

HEMORRHOIDS.—Hemorrhoids are considered to be a source of bleeding if blood is observed to come from them and more important lesions have been excluded.

SUMMARY

The manifold problems of gastrointestinal bleeding have been reviewed. Worthy of re-emphasis are:

1. Each physician should follow a course of management in keeping with his local hospital facilities.
2. If available, better results will be obtained by aggressive diagnosis and treatment of massive upper tract bleeding by a team of internist, surgeon and radiologist than by individually managed, nonoperative, nontransfusion programs.
3. Early x-ray diagnosis of previously undiagnosed massive bleeders is safe and very helpful.
4. Rare or unusual causes of bleeding from the entire gastrointestinal tract appear with sufficient frequency to require that physicians be alert to the unusual and the uncommon.

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